discomfort is pronounced it can be done or he may give up with the first feeling of effort.

To sustain the mercury column in the "fatigue" test requires some skill and a proper set of the lips may ease the performance so much that twenty and more seconds are added to the length of the hold. The expiratory force test, if well done, requires great effort and the height of the blow often depends upon the ability to coordinate the contraction of chest and abdominal muscles.

The coefficients of correlation for the power of breath-holding, vital capacity, "fatigue" test, expiratory force and extra respiratory reserve factor, with compensation to low oxygen, were found to be of such low value that the simple respiratory tests could not be accepted as aids in determining fitness for high altitude flying.

The degree of physical fitness it appears as shown by an examination of 31 temporarily unfit men cannot be measured either by the simple respiratory tests or by the low oxygen of the rebreathing test.

Seven men were able to improve their records in the respiratory tests by practice.

SACCULAR ANEURYSM OF THE DESCENDING THORACIC AORTA, WITH DIRECT RUPTURE INTO THE LOWER LOBE OF THE LEFT LUNG AND THE LEFT PLEURAL CAVITY:

THE REPORT OF A CASE AND REMARKS ON RUPTURE AND HEMOPTYSIS IN AORTIC ANEURYSM.

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At this time when quantitative and functional studies are so much in vogue, one hesitates on first thought to present a morphologic study. Nevertheless, the writer is of the opinion that such studies still hold a definite and valuable place in clinical medicine. In spite of the fact that our viewpoint has been expanded to incorporate the newer ideas of functional pathology, the clinician must still depend in a large part upon a thorough knowledge of gross pathology for his interpretation of physical findings. With this idea in mind it appears that unusual and instructive cases should be conscientiously studied and recorded, notwithstanding the fact that some of the work will doubtless be a repetition of what has been done.

The medical literature of aneurysm in all its phases is already astonishingly extensive and varied, and associated with its scientific development and collaboration are innumerable illustrious names in the annals of medicine.

The present paper was stimulated by a case which recently occurred in the Medical Service of the University of California Hospital, which the writer unfortunately did not see antemortem. The necropsy findings, however, directed my attention to the subject of aneurysm of the descending thoracic aorta, with special reference to rupture into pulmonary structures. The following report is interesting in the light of the necropsy findings:

Case History. The patient, J. S., a white man, aged fifty years, American, was admitted to the University of California Hospital on March 29, 1920, at 7 P.M. and died on March 31, 1920, at 2.30 P.M. On admission his temperature was 38.4° C., respiration 36 and pulse 104. He complained of "cough and weakness," which was of four years' duration, and of a pain in the left lower chest.

Family History. Father died at sixty-eight of unknown cause; mother died at seventy-two of old age; two brothers killed; one sister died at twenty-seven during childbirth. Patient married at twenty-seven and was divorced four years later; no children.

Past History. Born in Colorado and lived in California from thirty to fifty. He has been employed as a miner and steel-miller.

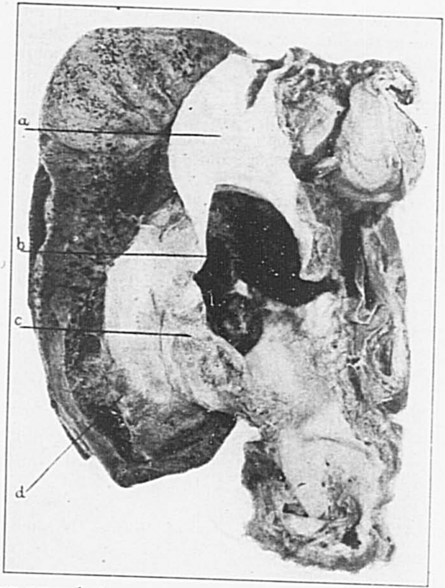
Diseases. Does not remember having childhood diseases. Influenza at twenty-five and again at forty-eight. He had pneumonia at thirty-four, and Neisser infection without known complications at eighteen and forty; lues denied.

Accidents. He is said to have fractured skull at twenty-three.

Habits. He had used alcohol steadily all his adult life and had been a heavy smoker.

Present Illness. Four years ago the patient had a sharp pain in the right lower chest, which increased in intensity during coughing and deep breathing. Since then he states that he catches cold very readily and has had a chronic cough; he has been below par most of the time. About four months ago the patient had a severe cold, with a chill and fever, but was not sick enough to be in bed. Since then he has improved slightly but has not felt as well as formerly. Four days ago patient was again seized with chills and fever, and yesterday morning on arising he felt a sharp pain in the lower left chest; this pain was more marked on deep breathing and coughing and was relieved by lying on the affected side. Although he has had some cough for the last four years, this has been more severe during the last three months. Recently he has had considerable sputum, mostly of the colorless, stringy, mucous variety but never blood-tinged. There is no history of hemoptysis nor night-sweats. His best weight is 180 pounds; average weight 170 pounds; present weight 158 pounds. He has had some shortness of breath off and on for the last three years, but this has been especially noticeable during the last three months.

Physical Examination. Well-developed and nourished man, lying on the left side, coughing frequently and expectorating a thin sputum mixed with a small amount of light greenish, mucopurulent material. Patient complains of sharp pain in the left lower chest. Head, ears and nose are negative. Eyes: Bilateral pterygium on the corneæ; pupils regular and equal and reacting actively to light and accommodation; extra-ocular movements well performed; fundi normal. Mouth: The teeth are absent; tongue heavily coated, symmetrical and freely movable. Tonsils: Atrophic; pharynx is injected. Neck: Negative and the thyroid is not palpable. Chest: Conforms to the moderate pigeon-shaped variety. Right side is more prominent than the left anteriorly. In the upper thoracic region the spine is slightly deviated to the right. The right supraclavicular area is depressed. There is a



Specimen removed at autopsy; posterior view. *a*, descending thoracic aorta; *b*, opening into the aneurysmal sac; *c*, posterior wall of the aneurysm; *d*, lower lobe of the left lung.

marked impairment of the percussion note in the left axilla which extends forward, fusing with the relative cardiac dulness and extending posteriorly to the left base. Over this area tactile fremitus is absent. There is a friction rub high in the left axilla. Anteriorly below the left clavicle the breath sounds are harsh and loud. Grocco's triangle of dulness is made out at the right base along the spine. Heart: P. M. I. is best heard at the fifth rib in the left parasternal line; the R. C. D. is displaced markedly to the right; the right border of the R. C. D. is made out 5.5 cm. to the right of the midsternal line in the fifth right interspace. There is a loud, blowing systolic murmur heard best at the aortic area. This murmur can be heard as far up as the first interspace and as low down as the third interspace on the right side. There is no diastolic shock over the sternum to be made out. The rhythm is regular; the rate is 108 per minute. Pulse shows good tension, rapid rise and fall, and the vessel wall is not palpable. Blood-pressure is $\frac{110}{80}$. Abdomen has normal contour and is rather resistant to deep palpation; no definite tenderness nor masses are made out. Genitalia are negative. Deep reflexes are equal and active throughout. The mental state is apparently normal. Blood examination: R. B. C., 3,800,000; W. B. C., 13,000; differential count (100 cells): P. M. N., 81; S. M., 14; L. M., 5; P. M. E., 0; P. M. B., 0. Urine examination: Sp. gr., 1.026; cloudy; acid; albumin, F. P. T.; sugar, 0; sediment, many urates and a few pus cells, but no casts. Impression: Tuberculous pleurisy, with effusion at the left base and displacement of the heart to the right.

March 31, 1920, at 9 A.M. Patient complaining of pain in the left side especially on cough. Side strapped with adhesive. Patient coughed considerably.

March 31. At 2.30 P.M. adhesive strapping was removed in preparation for thoracentesis. Patient continued coughing in paroxysms. He expectorated a small amount of a foamy, mucoid sputum and complained of pain in the left lower chest. Suddenly he began bringing up a small amount of blood, at the same time he became very restless and sat up straight in bed, and after four or five gasping spells he became unconscious and cyanotic, and respiration ceased rather sharply. The whole amount of blood expectorated did not exceed 30 or 40 c.c. The heart-beat was audible for a moment or two after respiration ceased.

Wassermann (on blood serum postmortem) was triple plus.

Fluoroscopic and roentgen-ray examinations had not been done because the patient was too ill to move.

Clinical impression as to the cause of death: Massive pulmonary hemorrhage into a bronchus in a case of ulcerative pulmonary tuberculosis.

Postmortem Record of the Pathologic Laboratory. A. 20.59, University of California, performed by Dr. G. Y. Rusk, autopsy one hour after death.

Anatomic Diagnosis. Aneurysm of the aorta, with rupture into the left lower lobe and into the left chest cavity; syphilitic aortitis; dislocation of the heart to the right apex in the midline; chronic local pleurisy about the aneurysm; pulmonary syphilis (?); chronic infarct in the spleen; acute anemia; chronic aortic valvulitis, with adhesions between the segments leading to slight stenosis.

Remains of a muscular, well-nourished male adult of normal stature. Rigor mortis absent. Body heat well retained. No superficial lymphadenopathies. The pupils are circular and equal. Moderate conjunctivitis is present. The left chest appears slightly fuller than the right.

On making the usual median incision the bodily fats are normally preserved and of normal color. The voluntary muscles are somewhat pale and the rib cartilages are somewhat more than normally calcified. On opening the left chest cavity there is a gush of blood-stained serum. The posterior surface of the sternum is negative. The left chest cavity contains an abundance of clotted blood and separated serum.

The heart is displaced to the right, so that the apex lies in the midline. The chest cavity is free from adhesions or fluid.

The right lung is voluminous and shows an average amount of pigmentation. It is hypercrepitant on palpation throughout. On section it is negative except for a moderate grade of diffuse emphysema.

The left lung, while somewhat compressed by the great amount of fluid in the pleural cavity, still is somewhat enlarged. The upper lobe is diffusely hypercrepitant, the lower lobe posteriorly superficially shows ragged point of rupture situated about one-third of the distance above the diaphragm on its posteromesial aspect. Adjacent to this, and extending to what subsequently proved to be an aneurysmal sac, is an area of diffuse opaque pleural thickening, but without adhesions. The tissues of the lower lobe, especially posteriorly, are suffused with hemorrhage which gradually diminishes in intensity toward the more anterior portions. The lung was removed together with the entire aorta, which will be described below. On section through the lower lobe there is found an area of irregular fibrosis extending in a wedge-shaped manner from near the root to the periphery through the center of the lower lobe. This area contains several patches of necrotic material and the process strongly suggests scarring associated with gummata.

The pericardial cavity contains about 30 c.c. of clear, fluid, serous transudate. The heart is of normal size considering the general development of the individual; it weighs 320 grams. The epicardial fat is normal. The relative size of the chambers appears normal. The endothelium of all the cavities, including the valves, except the aorta, appears normal. The myocardium is of normal thickness. The muscle is contracted. No areas of fibrosis were observed. The aortic valve shows the right posterior segments to be adherent and the line of juncture thickened, pulled foreward, separating the region of attachment of the segments for about 1 cm. from the aortic wall. On either side of this line of attachment there is a diffuse, gradual, lessened thickening of the adherent cusps, which extends to the region of the corpus on the posterior segment and throughout the whole of the right segment. This deformity appears to have led to a slight stenosis. The aorta just above the aortic cusps shows some irregular, soft, slightly yellowish superficial patches of atheroma, and in addition presents a number of areas, with irregular depressed, stellate creases of syphilitic aortitis. This condition of specific aortitis occurs with remarkable frequency throughout the whole length of the aorta. In the mid-thoracic region the left wall of the aorta presents a large opening, 8 x 10 cm., where it opens widely into an aneurysmal sac, approximately 8 cm. deep. The base of the aneurysm is formed by a thrombotic layer, which is supported on the thinned-out wall. The point of actual rupture into the bronchi is not determined until after fixation of the specimen.

The esophagus is normal, except that it was distinctly misplaced to the left by the aneurysmal dilatation.

The vertebra also as yet shows no destruction.

The spleen is moderately increased in size and somewhat softened. On section the pulp is pale, the Malpighian bodies are fairly numerous and only moderately distinct on the pale background. Corresponding to a small puckered scar in the cortex is a fibrous tissue thickening which extends about 3 mm. into the spleen, apparently the result of quite an old infarct.

The adrenals appear normal.

The kidneys appear normal.

The remainder of the urinogenital apparatus appears normal.

The pancreas shows a slight fibrosis in its distal half.

The liver appears normal except for slight palor.

The mesenteric and retroperitoneal lymph nodes are negative.

The muscosa of the gastro-intestinal tract appears normal.

Occurrence. Aneurysms of the descending thoracic aorta are the least common of all the aortic aneurysms, occurring only a little more than half as frequently as abdominal aneurysms. In 64 cases of aneurysms of the aorta among 2200 autopsies at the Johns Hopkins Hospital only 3 involved the descending thoracic aorta (Osler[1]). Since the publication of Osler's monograph[2] on this subject, in 1903, little has been added to our clinical knowledge of descending thoracic aneurysm; however, since then much scientific advance has been made as to the causal relationship of syphilis to vascular disease and aneurysm formation, refinements in the roentgen-ray study have been outlined and numerous methods of therapy have been proposed and advocated.

Diagnostic Difficulties. Osler pointed out that in general there were three groups of descending thoracic aneurysm based on clinical observation and physical findings liable to present serious difficulties in the diagnosis, namely: (1) Cases which present symptoms only and no physical signs or the signs are not characteristic; (2) cases in which the symptoms and signs suggest malignant growth; (3) cases in which there are obscure and puzzling chest symptoms suggestive neither of aneurysm nor tumor (the above case falls into the third group).

The features which warrant classification of this case in the third group are based on the following data: (1) The comparative latency of all clinical symptoms except the history of pain of three months' duration in the left lower chest; (2) the cough was never characteristic or typical of thoracic aneurysm; (3) the striking yet misleading physical signs indicating fluid at the left base; (4) the absence of hemoptysis except as a terminal event.

Anatomical Considerations. Owing to the protected and deep position of the descending thoracic aorta, extending from the sixth to the twelfth thoracic vertebra, aneurysms of this portion are frequently silent. It will be recalled that the descending arch passes backward and downward to the left of the trachea, behind the root of the left lung, to the left anterior aspect of the thoracic vertebra. As it passes downward the esophagus and thoracic duct lie on its right. The left pleura and lung lie to the left and anteriorly. The descending arch of the aorta and the thoracic aorta below are held firmly in place by the intercostal branches which are given off from the aorta. In view of the existence of these special anatomic relations distinctive clinical manifestations may be produced which offer evidence of deep-seated thoracic disease. There are, in general, two large groups of structures which appear to be involved in aneurysms of the descending thoracic aorta, and when involved may give clinical evidence of intrathoracic disease. From the clinical standpoint the first group is the most common, although many special examples of involvement of structures in the second group are found.

Group I. 1. Spine, vertebra and spinal nerves. 2. Lung, pleura and bronchi. 3. Trachea and esophagus.

Group II. 1. Pericardium and heart. 2. Other great thoracic vessels, including pulmonary arteries, veins, etc. 3. Stomach. 4. Thoracic duct. 5. Diaphragm.

It is not my intention to review the symptoms and signs produced by encroachment upon or rupture into all of the above structures but rather to point out some of the facts of interest in regard to ruptured descending thoracic aneurysms into the pulmonary structures and particularly into the lungs.

Rupture of Aneurysms in General. It is a well-known fact, particularly to pathologists who perform large numbers of postmortem examinations on medicolegal cases, that ruptured aneurysms stand high in the list of causes of sudden death. As long ago as 1895, Draper,[3] in this country, published a paper entitled "Sudden Death by Rupture of Thoracic Aneurysm Previously Unrecognized," which emphasizes this point.

In reference to the cause of death from aneurysm, Hare and Holder[4] state "that death occurs as a result either from rupture of the aneurysmal sac, from pressure of the sac upon important nerves and bloodvessels or from secondary changes which take place in these tissues and in other vital organs as a direct or indirect result of such pressure." Arnold[5] made a study of the cause of death in aneurysms of the aorta which do not rupture. He found in a large series of aortic aneurysms of all types 110 of the descending aorta, and of these 15 per cent. were unruptured at death, whereas 66 per cent. were found ruptured. From his statistics it also appears that descending aortic aneurysms are slightly more liable to rupture than those in other portions of the aorta.

Oswald Browne's figures indicate definitely that the most common termination of aneurysms of the descending thoracic aorta is rupture into the left pleural cavity. In his series of 17 cases in which 14 ruptured, 7 instances of rupture into the left pleural cavity occurred. The incidence of rupture at other points was as follows: Right pleural cavity, 2 cases; left bronchus, 1 case; esophagus, 3 cases; subserous tissue, 1 case.

Wilson[6] collected from the literature 75 cases of descending thoracic aneurysm, 53 of which had ruptured. In these ruptured cases he found that 13 cases had ruptured into the left pleura, 1 into the right pleura, 6 into the left lung, 16 into the left bronchus, 2 into the trachea, 2 into the pericardium and 9 into the esophagus. It was stated that in 2 instances rupture had occurred into the thoracic cavity, in 1 instance into the abdominal cavity, and once into the neck. In the remaining 22 cases rupture did not occur. Erosion of the vertebræ occurred in 26 instances, whereas an external tumor was encountered in only 6 of the 75 cases. In this series the clinical diagnosis was missed in 50 per cent. of the cases. In Osler's series of 14 cases, rupture into the pleura occurred 3 times, whereas in Wooley's[7] recent series of 9 ruptured aneurysms of all types 2 ruptured into the left pleural cavity. The foregoing figures emphasize the marked tendency that aneurysms of the descending thoracic aorta have for rupturing into the pulmonary structures, and particularly into the left pleural cavity.

Hemoptysis. Clarke,[8] in 1905, reported a remarkable case of thoracic aneurysm which had come under his observation. The case was unusual in that the patient had a series of 16 copious hemorrhages, and in a period of seven and a half weeks lost a total of fourteen pints of blood before exitus occurred with hemorrhage. In the last moments of life during the fatal hemorrhage the physical signs of fluid at the right base were made out. The autopsy revealed a saccular aneurysm of the ascending arch of the aorta projecting into and occupying a great portion of the upper lobe of the right lung, with rupture of the aneurysm into this lobe and extensive hemorrhage into the right pleural cavity. The striking feature presented by this case is the possibility of copious non-lethal hemorrhage in cases of thoracic aneurysm.

In the same paper Clarke refers to a case reported by Johnson[9] which appears very similar to the case I report above. Johnson's patient had long-standing symptoms of chronic pleurisy, but no history of hemorrhage until the fatal one. Aneurysm was not suspected, but at autopsy one was found of the descending aorta involving the lower left lobe of the lung. The aneurysmal walls were only one-quarter inch thick, composed of fibrin, and the lung and pleura were indurated.

Clarke concludes from his study of aneurysms in reference to hemoptysis "that hemoptysis is a common fatal termination in thoracic aneurysm, a very large hemorrhage may occur in this disease, without causing death of the patient, and that occasionally a patient may have one or more large hemoptyses, but finally die from some other complication of the aneurysm. The point of bleeding may be from rupture into the trachea or bronchi and may be stopped by the opening being plugged with fibrin or it may occur from involvement of the lung tissue itself. In the latter case it is probable that the small bronchioles open into the wall of the sac, but are compressed by it, that they normally remain closed, or are covered with fibrin. On exertion of moving, either the fibrin shifts its position or the straining of the coughing opens the ends of the small bronchi and the blood leaks out. When the patient again becomes quiet, either naturally or by means of narcotics the original condition is resumed and bleeding is temporarily arrested."

The foregoing statement appears to be a fairly reasonable explanation of the intermittent nature of hemoptysis in some of these cases.

Hall[10] in an able paper on the subject of intrathoracic aneurysm groups the mechanisms of pulmonary hemorrhage in these cases under three heads: (1) Direct hemorrhage, that is, when there is a leak from the aneurysmal sac into the air passages; (2) indirect hemorrhage, that is, when the hemorrhage is the result of pressure either on the air passages, the bloodvessels or the lung tissue itself; (3) hemorrhage secondary to cardiac disease with engorgement, of the pulmonary circulation.

Remarks on Other Symptoms—Pain, Cough and Dyspnea. In my case presented above pain played the conspicuous rôle in the symptomatology. Hewlett and Clarke[11] have pointed out in a small series of descending thoracic aneurysms that pain was the prominent symptom. They found it was usually of extraordinary severity, constant, subject to nocturnal exacerbations and resistant to all forms of medication. In general, pain is of three kinds: (1) Anginal, (2) that due to pressure, and (3) neuralgic.

It is a well-known fact that aneurysm often produces the clinical picture of angina pectoris,[12] and, further, that obscure and deep-seated thoracic pains of all variations are sometimes produced by pressure of intrathoracic aneurysmal tumors. In the neuralgic type pain reaches its maximum intensity and persists when the aneurysmal sac erodes the spine.

The mechanism of pain in thoracic aneurysm has been explained in various ways. Increased tension of the walls of the aorta can of itself become a cause of pain by stretching of the delicate nerves which run in the walls of the aorta producing a neuritis. Oliver[13] states that digitalis administered in suspected cases often augments pain in the chest, since by improving the force of contraction of the left ventricle and by constricting the peripheral arteries it raises arterial tension. Other factors in the production of pain include, of course, that caused by pressure on adjacent structures and the coexisting inflammation.

Cough and dyspnea are practically always constant features of involvement of the aortic arch, but are less constant in aneurysm of the descending portion of the aorta. In Osler's 14 cases these symptoms were present in 10 instances. Bronchorrhea and bronchiectasis result at times from pressure on the main bronchus.

Summary. A case of descending thoracic aneurysm is presented which had ruptured directly into the left lower lobe and left pleural cavity. The physical signs were misleading and the clinical diagnosis was incorrect. It is probable that if a roentgen-ray study had been possible it would have aided materially in determining the true nature of the pathologic process. Rupture into pulmonary structures of descending thoracic aneurysms is one of the most frequent occurrences, particularly rupture into the left pleural cavity, less commonly into both lung and pleural cavity. Hemoptysis must be regarded as a symptom of thoracic aneurysm as well as a symptom of other thoracic disease, such as tuberculosis, mitral stenosis and other mediastinal tumors. It may be frequent and persistent before a fatal hemorrhage occurs.

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SILVER SALVARSAN IN THE TREATMENT OF SYPHILIS.[*]

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Through the courtesy of Ehrlich's distinguished pupil and successor, Professor Kolle, early in November, 1919, Colonel Earl H. Bruns, chief surgeon, A. F. in G., received 1000 ampoules of silver salvarsan from the George Speyer Haus Laboratories, Frankfürt, Germany. Colonel Bruns turned this preparation over to Major Robert W. Kerr, Medical Corps, the Commanding Officer of the Base Hospital (now Station Hospital), A. F. in G., Coblenz, Germany, with instructions concerning its use which he had obtained personally from Professor Kolle. The first results seemed favorable and its further use was decided upon. Additional

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[*] Weichbrodt, Knauer, Seoli-Bonn and Gennerich: Ztschr. f. Aerztliche Fortbildung, Georg Speyer Haus, Frankfürt.